# Outcome of Patients Experiencing Cardiac Arrest With Carbon Monoxide Poisoning Treated With Hyperbaric Oxygen

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**Study objective:** We sought to examine the outcome of a subgroup of patients with extreme carbon monoxide (CO) poisoning, specifically those discovered in cardiac arrest, resuscitated, and subsequently treated with hyperbaric oxygen ($HBO_2$). Opinions of hyperbaric medicine physicians regarding the treatment of such patients were also sought.

**Methods:** Records of patients treated with $HBO_2$ for acute CO poisoning at Virginia Mason Medical Center in Seattle from September 1987 to August 2000 were reviewed. Those who were resuscitated from cardiac arrest in the field before $HBO_2$ treatment were selected for detailed analysis. Patient demographic data and information regarding circumstances of the poisoning, resuscitation, $HBO_2$ treatment, and subsequent course were extracted and collated. In addition, a postal survey of medical directors of North American $HBO_2$ treatment facilities regarding opinions about the management and outcome of such patients was performed.

**Results:** A total of 18 patients were treated with $HBO_2$ after resuscitation from CO-associated cardiac arrest. They included 10 female and 8 male patients ranging in age from 3 to 72 years. Sources of CO included house fires (10 patients) and automobile exhaust (8 patients). Patient carboxyhemoglobin levels averaged 31.7%±11.0% (mean±SD), and arterial pH averaged 7.14±0.19. Presenting cardiac rhythm was a bradydysrhythmia in 10 of 18 patients. $HBO_2$ treatment was administered an average of 4.3 hours after poisoning (≤3 hours in 10 patients and ≤6 hours in 15 patients). Despite this, all 18 patients died during their hospitalizations. Medical directors of hyperbaric treatment facilities estimated a 74% likelihood of survival for a hypothetical patient with this presentation.

**Conclusion:** In this consecutive case series, cardiac arrest complicating CO poisoning was uniformly fatal, despite administration of $HBO_2$ therapy after initial resuscitation. Survey results suggest that physician education regarding this subset

of CO-poisoned patients is needed. The prognosis of this condition should be considered when making triage and treatment decisions for patients poisoned to this severity.

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## INTRODUCTION

Carbon monoxide (CO) poisoning is a significant health problem in the United States. Although the exact incidence is unknown, one study estimated that more than 40,000 patients are seen annually in US emergency departments for recognized CO poisoning.[1] CO toxicity is mediated by a number of mechanisms,[2] with cardiac and central nervous system manifestations resulting in the most significant morbidity and mortality.

Hyperbaric oxygen ($HBO_2$) is used to treat some patients with CO poisoning. In 1992, an estimated 2,355 cases were treated with $HBO_2$ in the United States.[3] Only the most severely poisoned patients are typically selected for treatment with $HBO_2$.[3] The selection criteria recommended by the Undersea and Hyperbaric Medical Society include transient or prolonged unconsciousness, neurologic signs, or cardiovascular dysfunction.[4] The purpose of this study was to investigate the outcome of patients with the most severe form of cardiovascular dysfunction from CO poisoning, cardiac arrest, when treated with $HBO_2$ after successful initial resuscitation. In addition, a survey was performed to explore the attitudes of practicing hyperbaric medicine physicians regarding treatment of such patients.

## MATERIALS AND METHODS

A departmental log of patients administered $HBO_2$ therapy for acute CO poisoning at Virginia Mason Medical Center in Seattle from September 1, 1987, through August 30, 2000, was used to identify individuals treated with $HBO_2$ while mechanically ventilated. ED and hyperbaric department records of those patients were reviewed to identify patients resuscitated from cardiac arrest in the field before hyperbaric therapy. Cardiac arrest was defined for the purpose of this study as discovery of a patient with absence of a detectable pulse, unresponsiveness, and apnea. Data abstracted for such cases included demographic information; circumstances of CO exposure; details of resuscitation, transport, and $HBO_2$ treatment; and final outcome. Retrospective reviews of this type are exempt from institutional review board approval at our institution.

In addition, a postal survey was mailed in March 2000 to the medical directors of all 317 clinical hyperbaric chamber facilities in the United States, Canada, and Puerto Rico listed in the 1998 Undersea and Hyperbaric Medical Society chamber directory.[5] Medical directors were presented the following clinical scenario:

> A 35-year-old female is accidentally exposed to carbon monoxide from automobile exhaust and discovered unconscious on the garage floor. Paramedics find her pulseless with idioventricular rhythm, heart rate 20 per minute. Following resuscitation in the field, she is transported to your hospital's emergency department. She arrives there intubated, comatose, unresponsive to stimulation, in sinus tachycardia, with blood pressure 100/60. Arterial blood gas analysis demonstrates pH 7.16, $P_{CO_2}$ 29, $P_{O_2}$ 480, and COHb 30.7%. Resuscitation, transport, and emergency department stabilization have taken three hours.

A questionnaire asked whether they would recommend $HBO_2$ therapy for such a patient and asked them to estimate prognosis.

Simple descriptive statistics were used to report results.

## RESULTS

In the 13-year period studied, 980 patients were treated with $HBO_2$ for severe CO poisoning. Among these, 18 (1.84%) were treated with $HBO_2$ after they were resuscitated from cardiac arrest in the field, complicating their CO exposure. These included 10 female and 8 male patients ranging in age from 3 to 72 years (mean±SD, 35±17 years; Table 1).

Sources of CO included house fires (10 patients) and automobile exhaust (8 patients). Seven exposures were accidental, 7 were intentional, 1 was homicidal, and 3 were of undetermined intent. Six patients (4 intentional and 2 accidental exposures) were known from toxicology screening to have been associated with exposure to other neuroactive drugs, including ethanol (4 patients), opiates (3 patients), benzodiazepines (2 patients), and tetrahydrocannabinol (1 patient). It was not possible to determine duration of CO exposure from medical record review in most cases.

Each patient's cardiac rhythm at the time of discovery is listed in Table 1. In 10 of 18 cases, patients were found with a bradydysrhythmia, typically described in the medical records as an idioventricular rhythm. The duration of field resuscitation was recorded in available medical records for only 6 patients and ranged from 19 to 45 minutes.

On arrival in the initial ED, most patients (12/18) were described as comatose and unresponsive to stimulation. A minority were described as demonstrating movement in response to stimulation (3/18), some degree of respiratory effort (2/18), or sluggish pupillary response to light (1/18). Presenting carboxyhemoglobin level was available in all patients and ranged from 14.6% to 55.6% (mean±SD, 31.7%±11.0%). ED arterial blood gas values were available for 17 of 18 patients and were notable for severe metabolic acidosis, with an average pH of 7.14±0.19 (mean±SD). Cyanide levels were not obtained in any patients.

Patients were treated in a multiplace hyperbaric chamber while intubated and mechanically ventilated. $HBO_2$ treatment was initiated 2 to 14 hours after removal from CO exposure, with an average time to treatment of 4.3±2.8 hours (mean±SD). $HBO_2$ treatment was initiated 3 or fewer hours after discovery in 10 of 18 patients and after 6 or fewer hours in 15 patients. Patients were treated with one of 2 $HBO_2$ protocols. Six patients treated from 1987 through 1992 received four 23-minute periods of 100% oxygen breathing at 2.8 atmospheres absolute (atm abs) pressure. Twelve patients referred after 1992 were treated with the US Air Force CO protocol.[6] This includes two 23-minute oxygen breathing periods at 3.0 atm abs, followed by two 25-minute oxygen periods at 2.0 atm abs. One patient's $HBO_2$ treatment was aborted after the initial oxygen period at 3.0 atm abs because of hemodynamic instability. Two of the patients treated with the US Air Force protocol received an additional $HBO_2$ treatment on the second day of hospitalization. Arterial blood gas measurements were obtained for 17 patients during $HBO_2$ treatment. Arterial $Po_2$ greater than 760 mm Hg was documented in 15 of 17 patients during $HBO_2$ therapy. In the remaining 2 patients, acute lung injury caused by smoke inhalation was believed to be the cause for impaired oxygenation.

All 18 patients died during hospitalization (95% CI for the 0% survival rate, 0% to 18.5%). Time from discovery to death ranged from 9 hours to 7 days (median, 42 hours; 25th percentile, 24 hours; 75th percentile, 73 hours). In most cases, medical support was withdrawn after neurology consultation indicated severe, irreversible brain damage. Autopsies were performed in 17 of 18 cases. Causes of death recorded on death certificates by the county medical examiner were "hypoxic encephalopathy" or "anoxic encephalopathy" caused by CO poisoning in all cases.

Results of the survey of medical directors of North American hyperbaric oxygen treatment facilities are shown in Table 2. Responses were received from 150 (47%) facilities. When presented with a description of a case of CO poisoning typical of that seen in this series, 100% of responding medical directors would recommend treatment with $HBO_2$. They predicted 74% probability of survival after such treatment, with a 28% chance for complete recovery without neurologic sequelae.

**Table 1.**
*Patient data.*

| Patient No. | Sex | Age (y) | Initial Cardiac Rhythm | COHb | ED ABG (pH; $Pco_2$; $Po_2$) | Outcome |
|---|---|---|---|---|---|---|
| 1 | M | 72 | Bradydysrhythmia | 22.0 | Not available | Died |
| 2 | M | 41 | Bradydysrhythmia | 24.9 | 7.09; 23; 507 | Died |
| 3 | F | 45 | Bradydysrhythmia | 23.8 | 7.13; 20; 474 | Died |
| 4 | F | 45 | Asystole | 14.6 | 6.92; 15; 486 | Died |
| 5 | F | 49 | Bradydysrhythmia | 20.5 | 7.45; 23; 533 | Died |
| 6 | F | 5 | Bradydysrhythmia | 34.0 | 7.08; 31; 275 | Died |
| 7 | F | 47 | Ventricular fibrillation | 44.6 | 7.40; 21; 385 | Died |
| 8 | M | 37 | Asystole | 34.6 | 6.98; 47; 371 | Died |
| 9 | M | 31 | Bradydysrhythmia | 28.7 | 7.14; 37; 391 | Died |
| 10 | F | 41 | Bradydysrhythmia | 29.0 | 7.36; 23; 554 | Died |
| 11 | F | 34 | Ventricular fibrillation | 28.8 | 7.30; 29; 502 | Died |
| 12 | M | 3 | Unknown (pulseless) | 16.5 | 7.10; 14; 662 | Died |
| 13 | M | 37 | Bradydysrhythmia | 55.6 | 7.12; 22; 457 | Died |
| 14 | F | 47 | Ventricular fibrillation | 34.9 | 6.99; 39; 532 | Died |
| 15 | M | 5 | Bradydysrhythmia | 42.0 | 7.24; 37; 381 | Died |
| 16 | F | 31 | Asystole | 31.0 | 7.23; 36; 510 | Died |
| 17 | M | 28 | Asystole | 35.7 | 7.09; 44; 662 | Died |
| 18 | F | 33 | Bradydysrhythmia | 49.5 | 6.70; 27; 154 | Died |

**COHb**, Blood carboxyhemoglobin level; **ABG**, arterial blood gas analysis.

## DISCUSSION

Cardiac arrest complicating CO poisoning resulted in 100% mortality in this series, despite initial resuscitation

**Table 2.**
*Opinions of medical directors of North American hyperbaric oxygen treatment facilities regarding management and outcome of patients with CO-associated cardiac arrest (see Methods section for hypothetical case description).*

| | |
|---|---|
| Would you recommend $HBO_2$ treatment for this patient? | Yes 100% No 0% |
| If treated with $HBO_2$, what likelihood would you predict for the following 3 possible outcomes? | |
| 1. Death during hospitalization | 26%±23% |
| 2. Survival to hospital discharge with permanent neurologic sequelae | 46%±23% |
| 3. Survival to hospital discharge with eventual complete recovery | 28%±27% |

Values are given as mean±SD where applicable.

in the field with return of spontaneous circulation and subsequent $HBO_2$ treatment.

A number of studies have documented the poor prognosis of patients with out-of-hospital cardiac arrest in general. Rates of survival to hospital discharge in reported series have ranged from 1.4% to 26%[7-10] and are estimated to average 20% overall.[11] When one examines only those patients who achieve return of spontaneous circulation with resuscitation and are admitted to a hospital, survival rates in the range of 14% to 57% are reported.[7,9,10] A noncardiac cause for arrest has been identified with a poorer prognosis,[12] a factor relevant to all the patients in the present series. Discovery of the arrested patient in asystole or bradydysrhythmia has also been described as associated with a worse prognosis.[7,10] Among the 18 present patients, 4 were discovered in asystole and 10 in bradydysrhythmia.

Several published clinical series have included patients with CO-associated cardiac arrest. Among 5 pediatric patients with cardiac arrest caused by smoke inhalation reported by Suominen et al,[13] none survived. In a series of children with CO poisoning reported by Meert et al,[14] 10 patients were described who presented with cardiopulmonary arrest. Eight died with hypoxic-ischemic encephalopathy, and 2 survived, both with adverse neurologic sequelae. Hantson et al[15] described 10 adult patients admitted to an ICU in Paris, France, with cardiac arrest caused by smoke inhalation, all of whom died. All exhibited high blood carboxyhemoglobin and cyanide levels and died in the critical care unit of "brain failure." A report from Australia on 100 consecutive hospital admissions of adults with CO poisoning mentions that 5 presented with cardiac arrest.[16] The article implies that some may have survived but does not provide treatment information or morbidity data for that subgroup of patients. A large study from Helsinki, Finland, examining out-of-hospital cardiac arrests in that city from 1994-1995 included 5 patients with arrest caused by CO poisoning.[17] Personal communication with the first author of that study reveals that 3 of the 5 died in the field. The other 2 achieved return of spontaneous circulation but died in the hospital. Neither were treated with $HBO_2$. Their emergency medical system records include an additional 7 patients with CO-associated cardiac arrest in 1993 and 1996-1998, none of whom survived despite return of spontaneous circulation in 2 (M. Kuisma, personal communication, December 13, 1999).

Four previously published series include patients with CO-associated cardiac arrest who were resuscitated and treated with $HBO_2$. In 2 of the studies, a total of 11 such patients were reported, all of whom died despite $HBO_2$ therapy.[18,19] The third study described 23 patients treated with $HBO_2$ after resuscitation from CO-associated cardiac arrest.[20] Seventeen of the 23 died during the hospitalization, 6 of recurrent fatal cardiopulmonary arrests and 11 after the removal of life support systems after diagnosis of brain death. The ultimate outcome of the other 6 patients, including the degree of neurologic recovery, is unknown. The fourth study included 2 patients with cardiac arrest.[21] A patient treated with $HBO_2$ died, and one treated with normobaric oxygen survived.

It can be seen that cardiac arrest complicating CO poisoning carries a dismal prognosis, even if resuscitation accomplishes return of spontaneous circulation and the patient receives $HBO_2$ therapy. A number of reasons may be postulated to explain this. First, one must consider the possibility that $HBO_2$ is in some way detrimental in this group of patients. This seems unlikely in light of the fact that 4 of 6 prospective, randomized clinical trials reported to date comparing normobaric oxygen and $HBO_2$ treatment for acute CO poisoning of lessor severity have demonstrated benefit from $HBO_2$.[21-26]

A second possible explanation for poor prognosis might be inadequate $HBO_2$ treatment. The $HBO_2$ protocols that were used to treat our patients are standard therapy in the United States.[6] Whether additional repetitive $HBO_2$ treatments would have altered outcome is unknown. Although some previously published uncontrolled case series have proposed that repetitive treatment of CO-poisoned patients may be beneficial,[16,27] no prospective controlled data have demonstrated a clear benefit of repeat $HBO_2$ therapy over a single treatment.

It has been previously shown that $HBO_2$ treatment of CO-poisoned individuals is more effective when delivered within 6 hours of removal from CO exposure.[28] In the present series, more than half of the patients were treated within 3 hours, yet all died. The possibility that even more immediate $HBO_2$ therapy might be effective exists but seems unlikely.

Another explanation for the high mortality in this subgroup of CO-poisoned patients might be concomitant poisoning with other toxins, particularly cyanide. Among patients poisoned with CO in house fires, many would be expected to have simultaneous cyanide poisoning. This was documented in 100% of patients undergoing cardiac arrest sustaining CO poisoning from smoke inhalation in one report.[15] This does not, however, entirely explain the poor prognosis seen in the present series. Among the 18 fatal poisonings, 8 patients were exposed to CO from automobile exhaust, none of whom

would be expected to have simultaneous cyanide poisoning.

Finally, the poor prognosis may simply be because of the severity of the central nervous system insult that occurs when cardiac arrest complicates CO poisoning. CO toxicity is mediated in part through effects on hemoglobin, which are commonly known as "CO hypoxia."[2] A variety of other mechanisms have also been described. In patients who experience cardiac arrest, the nervous tissue insult is obviously compounded by the absence of oxygen delivery, which occurs when cardiac output ceases. Many of the other biochemical mechanisms of CO toxicity may be amplified by such ischemia. For example, if brain anoxia develops during CO exposure, cytochrome oxidase becomes reduced and CO binds to it without having to compete with oxygen. After the oxygen supply is replenished, the rate of respiration in CO-inhibited mitochondria is restored more slowly than in mitochondria that were exposed to oxygen deprivation alone.[2,29] In another animal model, a period of hypotension was required in addition to CO exposure for CO-mediated brain lipid peroxidation to occur.[30] It would appear that the combination of cardiac arrest with CO poisoning may present an overwhelming insult that is only rarely survivable.

Some limitations of our study should be noted. First, it was a retrospective review without uniform diagnostic entry criteria. As such, it is possible that additional patients referred and treated with $HBO_2$ also experienced cardiac arrest that was not recorded in their records because it was a less severe event. Such cases could have been missed by our review and might have had a better outcome. The study is also limited by its small size (18 patients). If the next 2 patients with the syndrome were to survive, the survival rate would be 10% and not 0%. A larger study is needed to conclude that $HBO_2$ therapy for such patients is futile.

All hyperbaric facility medical directors responding to our survey would treat such patients with $HBO_2$ if they had experienced return of spontaneous circulation with resuscitation and were in their own hospital's ED. This likely relates in part to the fact that no prior studies have been published reporting the dismal outcome of this subgroup of patients in a systematic manner and indicates that physician education is needed. It should be noted, however, that patients in our study did not present initially to our hospital's ED and were referred from an outside ED. This added a component of delay to hyperbaric treatment, and the remote possibility cannot be excluded that $HBO_2$ therapy delivered even earlier than in this study might be more effective.

On the basis of the present experience and review of the available literature, it must be concluded that cardiac arrest associated with CO poisoning carries an extremely poor prognosis for survival, with or without $HBO_2$ treatment. This should be taken into consideration when triaging such patients, especially if $HBO_2$ therapy requires significant transport of these critically ill patients.

Author contributions: NBH conceived the study. NBH and JLZ designed the trial. JLZ performed chart review for data extraction and database entry. NBH performed data analysis and drafted the manuscript. Both authors contributed significantly to its revision. NBH takes responsibility for the paper as a whole.

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